Vocal Cord Dysfunction
Case Study and Commentary, Michael A. O’Connell, MD

Vocal cord dysfunction (VCD) can be characterized as an abnormal adduction of the vocal cords during the respiratory cycle, particularly during inspiration [1]. The pathophysiologic mechanism by which abnormal vocal cord adduction occurs is unknown, although it has recently been speculated to be the result of altered autonomic balance [2]. The vocal cord abnormality is not felt to be factitious and typically cannot be reproduced by the patient voluntarily [1,3]. The end result of this abnormal vocal cord adduction is airflow obstruction at the level of the larynx and the creation of a sensation of dyspnea and chest tightness that can mimic asthma [1,3]. Because dyspnea and chest tightness are symptoms associated with systemic allergic reactions, VCD has also masqueraded as anaphylaxis with symptoms limited to the airway [4,5]. Further complicating the issue is that asthma and VCD can coexist in the same patient [4]; this scenario is difficult to unravel even by an experienced specialist. A published case series from a specialty respiratory hospital showed that VCD coexisted with asthma in 56% of their patients [6].

The published literature describing VCD consists primarily of case reports and case series. Broad epidemiologic studies and randomized controlled trials of various diagnostic and treatment strategies do not exist. Thus, the true incidence and prevalence of VCD is unknown, but it is believed to be more common than has previously been recognized. A recent abstract found that the published medical literature contains reports on 1458 patients with VCD [7]. Roughly 80% were adults and 20% were children [7]. There was a 3:1 female-to-male predominance, and the mean patient age was 40.5 years, with an age range from infancy to 82 years [7]. Early case series suggested a high incidence of associated psychiatric conditions, such as obsessive-compulsive disorder and neuroses/conversion disorders induced by childhood sexual abuse [6]. This unfortunately had the effect of labeling many patients as malingerers without organic illness [3]. Recently, several reports have highlighted other contributing etiologies, including gastroesophageal reflux [8,9], copious postnasal drip with chronic throat clearing [4],

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CASE STUDY

Initial Presentation

A 25-year-old woman presents as a new patient to a primary care physician with a 3-month history of episodic dyspnea and a choking sensation in her throat. These episodes are not associated with exercise, do not occur nocturnally, and are of sudden onset. She thinks that some of these episodes may have been triggered by exposure to airborne irritants, such as strong perfume or cigarette smoke.

History

The patient reports that she was initially seen in an acute care clinic and was told she had new-onset asthma. Faint wheezing was heard on physical examination of the chest. The patient was given an albuterol inhaler and told to use it when she felt short of breath, but the patient states that the inhaler is of no benefit during an attack. Her most recent episode was more severe, and she presented to a local emergency department (ED) where she was given nebulized albuterol and intravenous corticosteroids with gradual improvement over time. Spirometry was attempted in the ED, but the patient was unable to perform an adequate forced expiratory maneuver. Posterior-anterior and lateral chest radiography was performed and was normal. She was discharged with a diagnosis of poorly controlled asthma and prescribed inhaled corticosteroids on a daily basis. Despite the regular use of inhaled corticosteroids, the patient has continued to have episodes of dyspnea. After severe episodes, she often notices some hoarseness and intermittent dry cough. She denies a history of asthma in childhood and has never had similar episodes previously. She states that she has had significant postnasal drip recently, and her husband complains that she constantly clears her throat. Upon detailed questioning, she denies symptoms consistent with gastroesophageal reflux disease.

Past medical history is unremarkable. The patient was in good health and was taking no regular medications prior to the onset of these episodes of dyspnea. Family history is negative for atopic diseases and asthma. The patient is a non-smoker and drinks alcohol rarely. She has no pets in her home and is not regularly exposed to environmental tobacco smoke. Environmental history is unremarkable.

Physical Examination and Laboratory Testing

On physical examination, vital signs are normal and the patient is in no acute distress. Nasal mucosa is pale and boggy, and there is increased clear mucus in the nasal vault bilaterally. Lungs are clear to auscultation and percussion, and auscultation of the neck is clear without wheezing or stridor. Skin examination is normal without rashes or findings consistent with eczema. The remainder of the physical examination is normal.

Spirometry is performed in the primary care office and is interpreted as normal, with a forced vital capacity (FVC) of 97% of predicted, a forced expiratory volume in 1 second (FEV₁) of 96% of predicted, and a normal FEV₁/FVC. An inspiratory flow-volume loop is not performed.

Initial Diagnosis

Based on the history and clinical evaluation, the patient is diagnosed with chronic rhinitis complicated by mucus hypersecretion and labile asthma. She is started on a topical nasal corticosteroid spray and a long-acting β agonist; in addition, an oral leukotriene antagonist is added to her regimen of daily inhaled corticosteroids.

- What is the approach to diagnostic evaluation in a patient who presents with asthma-like symptoms but who does not respond to initial treatment?

Diagnostic Workup

These challenging patients should first be more thoroughly evaluated to ensure that the clinical diagnosis of asthma can be objectively confirmed by demonstrating the presence of reversible airflow obstruction and/or bronchial hyperresponsiveness [12,13]. The presence of reversible airflow obstruction can be demonstrated by showing greater than 20% variability in a series of home peak flow measurements or by obtaining spirometry during an acute episode that demonstrates airflow obstruction and confirming reversibility by subsequent administration of an inhaled bronchodilator with resultant 12% or greater improvement in FEV₁. Demonstrating the
VOCAL CORD DYSFUNCTION

Table. Presenting Features That Suggest a Diagnosis of Vocal Cord Dysfunction (versus Asthma)

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<th>History</th>
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<td>Sudden onset of severe dyspnea</td>
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<td>Sensation of choking or tightness in the throat</td>
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<td>Absence of nocturnal awakening with dyspnea</td>
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<td>Poor response to treatment with standard anti-asthma medications</td>
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<td>Requirement for corticosteroids (and often overuse)</td>
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<td>Exquisite sensitivity to occupational and environmental irritant triggers</td>
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<td>Multiple emergency department visits</td>
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<th>Physical examination</th>
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<td>Wheezing or stridor heard on auscultation over the larynx</td>
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<th>Objective measurements of pulmonary function</th>
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<td>Normal (nonobstructive) expiratory flows (FEV₁ and FEV₁/FVC)</td>
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<td>Normal-appearing expiratory flow-volume loop</td>
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<td>Abnormal-appearing inspiratory flow-volume loop (attenuation, truncation)</td>
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FEV₁ = forced expiratory volume in 1 second; FVC = forced vital capacity.

The presence of bronchial hyperresponsiveness requires broncho-provocation challenge testing with a standardized agent such as methacholine. If reversible airflow obstruction and/or bronchial hyperresponsiveness cannot be confirmed, alternative diagnoses such as VCD should be considered [12,13].

Similar to the diagnosis of asthma, the diagnosis of VCD is not based on a single test or measure but rather the synthesis of information gathered from the history, physical examination, response to previous therapeutic interventions, and diagnostic testing [1,3,4]. One way to approach the diagnosis of VCD is to compare the clinical history and examination with that of asthma, highlighting the features of the presenting patient that are not consistent with asthma. There are several historical features that suggest VCD and are not typically associated with asthma (Table). These include sudden onset of severe dyspnea, localization of the tightness/constriction to the throat rather than the chest, and lack of symptoms nocturnally [3,4,11]. Most asthmatics have a more gradual onset of dyspnea and wheezing, and exercise and cold-air exposure are classic triggers. Even more specific for VCD is the lack of nocturnal awakening with dyspnea, which is one of the cardinal features of true asthma. More than 2 episodes of nocturnal awakening with dyspnea per month is one of the criteria in the U.S. National Asthma Education and Prevention Program asthma guidelines for poor asthma control [12]. VCD patients rarely, if ever, awaken from sleep with symptomatic VCD [3,11]. Additionally, personal and/or family history of atopy is strongly associated with asthma and is not as frequently associated with VCD [15]. In our institution’s experience, one of the key historical factors that is predictive of a subsequent diagnosis of VCD is the lack of response to appropriate asthma therapy. Patients who state that administration of β agonists and systemic corticosteroids provides no symptomatic benefit are unlikely to have asthma unless they are noncompliant or administering the medications using ineffective delivery technique. In light of this, if a patient previously reliably diagnosed with asthma subsequently develops VCD, the usual presentation is a marked change in control of respiratory symptoms with new acute and often severe episodes refractory to previously effective asthma medications [11].

During the physical examination, close attention should be paid to the examination of the upper and lower respiratory tract. The presence of wheezing heard loudest in the chest, chest hyperexpansion, and a prolonged expiratory phase during acute symptoms supports the diagnosis of asthma [12,13], while wheezing or stridor heard loudest over the neck and radiating into the chest suggests a laryngeal abnormality [11]. Asking the symptomatic patient a question such as “Where do you feel your shortness of breath is localized?” may be helpful because some VCD patients will point to their throat whereas asthmatic patients rarely will. Because both asthma and VCD are episodic disorders, the absence of physical examination findings in an asymptomatic patient does not exclude either diagnosis.

In terms of objective diagnostic testing, chest radiography is of little value in either diagnosing or distinguishing asthma from VCD, as chest radiographs may be normal in both conditions even during acute symptoms. Spirometry is the gold standard for the diagnosis of obstructive lung disease such as asthma because it provides objective measurement of lung function and can demonstrate the presence of airflow limitation and reversibility with bronchodilator medication [12,13]. Spirometry is also helpful in the diagnosis of VCD. Typically, there is no evidence of expiratory airflow limitation (ie, FVC, FEV₁, and FEV₁/FVC are normal), even during an acute episode [3,4]. If the FVC and FEV₁ are reduced, they are typically reduced in parallel and the FEV₁/FVC is therefore normal, which is not consistent with airflow limitation obstruction. The most useful diagnostic feature relative to VCD available from more sophisticated spirometers is the ability to perform an inspiratory flow-volume loop in addition to the expiratory maneuver commonly obtained. Since VCD is characterized by adduction of the vocal cords during inspiration, the inspiratory flow-volume loop is classically truncated or attenuated [1,4] (Figure 1). Once again, a normal inspiratory flow-volume loop when the patient is asymptomatic does not exclude the diagnosis of VCD. Occasionally, patients may show other abnormalities such as blunting of the expiratory flow-volume loop or striking loop-to-loop variability between efforts [3].

Unfortunately, for many primary care physicians, spirometry is not readily available for a variety of reasons. Even if a
basic spirometer is available, the equipment may not be capable of performing an inspiratory flow-volume loop and/or the clinic and physician staff may not be trained in the proper performance or interpretation of inspiratory maneuvers.

• Can VCD present as refractory exercise-induced dyspnea?

There are several case reports in teenagers and young adults of VCD mimicking exercise-induced bronchospasm, especially in deconditioned athletes [14] and military recruits [15] forced to participate in vigorous exercise. However, VCD has even been reported in elite athletes [16]. Characteristic clinical features include symptoms occurring only with exercise and lack of response to exercise pretreatment with a β agonist [16]. Baseline spirometry is typically normal and exercise challenges may provoke symptoms without a corresponding drop in FEV₁ and FEV₁/FVC, essentially excluding the diagnosis of exercise-induced asthma.

Follow-up

Despite the aggressive anti-asthma therapy, the patient’s episodes continue and she becomes worried that she has developed “the world’s worst asthma.” Her primary care physician subsequently requests a consultation from an allergist-immunologist for comanagement of the patient’s difficult-to-treat asthma. Spirometry is repeated by the allergist while the patient is asymptomatic and once again the FVC, FEV₁, and FEV₁/FVC are normal. However, an inspiratory loop is obtained while the patient is asymptomatic and it is normal as well.

• What findings in this patient suggest a diagnosis of VCD?

There are several important findings from the patient’s history that suggest a diagnosis of VCD: sudden onset of acute, severe dyspnea; lack of response to aggressive anti-asthma pharmacotherapy including β agonists and corticosteroids; and the sensation of throat closing/choking during severe episodes. The patient has never complained of a nocturnal episode of dyspnea awakening her from sleep. Her history of recent onset of copious postnasal drip and chronic throat clearing suggests a possible triggering factor for laryngeal dysfunction in that chronic throat clearing can become an involuntary maneuver that results in tightening of the laryngeal apparatus. The cough and hoarseness after episodes are also suggestive of laryngeal hyperresponsiveness [17]. In terms of objective findings, spirometry has never demonstrated airflow limitation/
obstruction consistent with asthma although admittedly a spirometric examination has never been obtained during acute symptoms.

• What additional diagnostic tests are performed by a specialist when VCD is suspected?

The gold standard for confirming the diagnosis of VCD is direct visualization of the paradoxical vocal cord adduction during symptoms using fiberoptic rhinolaryngoscopy [1,3,4,11]. Additionally, many experts in the field prefer to perform a bronchoprovocation challenge with methacholine prior to the laryngoscopy to assess for the presence or absence of bronchial hyperresponsiveness [4,15]. In addition to confirming or disproving the diagnosis of asthma, methacholine challenge may provoke acute symptoms, which increase the diagnostic yield of the fiberoptic rhinolaryngoscopy. At our institution, we are prepared to perform the fiberoptic laryngoscopy immediately if the patient develops symptoms during the performance of the methacholine challenge.

Specialists who have access to and can perform fiberoptic rhinolaryngoscopy include allergist-immunologists and otolaryngologists, and methacholine challenges can be performed by pulmonologists and allergist-immunologists. In both private practice and many institutional settings, these various specialists often work in concert to evaluate the patient. If the patient’s initial diagnosis is refractory asthma, initial consultation by an asthma specialist is the best course of action, as consultation by an asthma specialist has been shown to be cost-effective in patients with atypical signs and symptoms and complicating diagnoses such as VCD [18].

Additional Follow-up

The allergist informs the patient that he suspects that she may not have asthma at all and he would like to do further testing. She undergoes a methacholine challenge that is negative for bronchial hyperresponsiveness, effectively ruling out the diagnosis of asthma and explaining why she has not responded to treatment with asthma medications. During the initial phase of the methacholine challenge, the patient is asymptomatic with normal inspiratory flow-volume loops (Figure 2A). However, in the last stage of the methacholine challenge, she begins to complain of her typical dyspnea symptom pattern. Physical examination reveals wheezing...
loudest in the neck area and faintly transmitted to the chest. Pulse oximetry on room air is 93%. Spirometry shows a normal expiratory flow-volume loop but a markedly truncated inspiratory flow-volume loop consistent with acute extrathoracic airway obstruction (Figure 2B). The patient is immediately prepped and consents to fiberoptic rhinolaryngoscopy, which confirms striking vocal cord adduction during inspiration in contrast to what would be expected during normal inspiration (Figure 3). In addition, copious clear mucus is seen in the posterior nasal vault draining into the pharynx. The patient is shown a video of her laryngeal examination and she seems relieved that an abnormality has been found that can explain her refractory symptoms.

• What is the approach to treatment for VCD?

No randomized controlled trials are currently published comparing treatment protocols for VCD patients. Most experts take a 2-pronged approach to treatment, involving treating underlying triggering and exacerbating factors plus speech therapy with the goal of teaching the patient voluntary maneuvers that will abort an acute attack [4,11]. For example, underlying gastroesophageal reflux if present should be treated with appropriate antireflux medications and lifestyle modification, and any confirmed psychiatric disorders should be treated with appropriate pharmacotherapy and counseling. Speech therapy classically involves breathing exercises and laryngeal relaxation techniques that require referral to an experienced speech language pathologist [19–21]. In many instances, speech language pathologists can also play an important role in the diagnosis of VCD, especially if the patient is complaining of associated cough and/or hoarseness. Because of the complexity of this disorder, a multidisciplinary approach is often needed to address all contributing factors [21]. Pharmacotherapy is typically not indicated or helpful [1,3,4,11]. Unless the patient has asthma coexisting with the VCD, asthma medications should be discontinued (often by tapering), in part to reinforce that the patient does not have asthma.

Initiation of Therapy

After completion of the methacholine challenge and fiberoptic rhinolaryngoscopy, the allergist reinforces the message that the patient does not have asthma and recommends that she can safely taper the anti-asthma therapies until they are discontinued. The allergist further recommends a referral to a speech language pathologist for initiation of training in appropriate breathing and laryngeal relaxation exercises. Because copious postnasal drip is likely playing a contributing role by increasing laryngeal tone as a result of chronic throat clearing, the patient is instructed to increase her oral fluid intake and perform daily nasal irrigation with normal saline.

Additional Follow-up

At a 6-week follow-up visit with the allergist, the patient reports that she has had 3 visits with a speech language pathologist and she is beginning to master the breathing and laryngeal relaxation exercises. She has noticed a dramatic reduction in the amount of postnasal secretions with the use of saline nasal irrigation. She has not had any severe episodes of dyspnea even though she has stopped all her anti-asthma medications. She has had several mild episodes of dyspnea that she was able to treat using her breathing and laryngeal relaxation techniques.
maneuvers. She expresses that she feels she can now successfully take control of her problem because she knows what is wrong with her and how to take care of it. Previously, she admitted that she was quite anxious because of the uncertainty regarding her diagnosis and the lack of response to aggressive treatment. Because of the success of the above interventions, she is returned to the care of her primary care physician with recommendations for continuation of her speech therapy with follow-up as determined by the speech language pathologist and follow-up with the allergist in 6 months.

- What is the prognosis for patients with VCD?

Once again, formal outcome measures from longitudinal controlled trials using standardized treatment protocols for VCD are not published in the medical literature. From published case series, the overall prognosis for VCD patients appears favorable [1,22]. In our experience, factors that predict a favorable outcome include the availability of an experienced speech therapist, a compliant patient willing to accept the diagnosis and take steps to control the disease process, and the absence of major psychiatric illness.

CONCLUSION

As asthma continues to increase in frequency and severity, more complex patients are presenting to primary care physicians. VCD should be considered as a masquerader of asthma in patients with atypical presenting symptoms that are refractory to standard anti-asthma medications. Referral to an asthma specialist can facilitate proper diagnosis and management, which should result in optimal control of the patient’s respiratory symptoms and avoidance of costly, unnecessary, and potentially harmful medical interventions including chronic overuse of corticosteroids.

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CME EVALUATION: Vocal Cord Dysfunction

DIRECTIONS: Each of the questions below is followed by 4 possible answers. Select the ONE lettered answer that is BEST in each case and circle the corresponding letter on the answer sheet.

1. All of the following are commonly seen in vocal cord dysfunction EXCEPT
   (A) Wheezing  
   (B) Dyspnea  
   (C) Hypoxemia  
   (D) Normal chest radiograph

2. Which of the following is the “gold standard” for diagnosing vocal cord dysfunction?
   (A) A positive methacholine challenge test  
   (B) Spirometry showing a forced expiratory volume in 1 second of less than 70% of predicted  
   (C) Chest radiograph showing flattened diaphragm  
   (D) Direct visualization of paradoxical vocal cord adduction during acute symptoms using fiberoptic rhinolaryngoscopy

3. All of the following are helpful in differentiating vocal cord dysfunction from asthma EXCEPT
   (A) Normal chest radiograph  
   (B) Presence of sensation of choking or tightness in the throat  
   (C) Absence of nocturnal awakening with dyspnea  
   (D) Poor response to treatment with standard anti-asthma therapies

4. Which of the following is NOT true of vocal cord dysfunction?
   (A) It is usually not factitious and cannot be voluntarily reproduced  
   (B) A positive methacholine challenge rules out the diagnosis  
   (C) It may coexist with asthma  
   (D) It can mimic exercise-induced asthma in teenagers and military recruits

5. Contributing etiologies in the pathophysiology of vocal cord dysfunction include:
   (A) Gastroesophageal reflux  
   (B) Postnasal drip with chronic throat clearing  
   (C) Psychiatric disorders associated with childhood sexual abuse  
   (D) All of the above
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2. A B C D
3. A B C D
4. A B C D
5. A B C D

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   _ Very clear  _ Somewhat clear  _ Not at all clear

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